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# Severe delirium on a background of Alzheimer's dementia – A devastating acute illness; report of a case

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## ABSTRACT

Delirium is an acute and reversible condition that is common in hospitalised patients. Patients with delirium have extended lengths of stay, double the mortality of matched controls and an increased risk of permanent cognitive decline. We present the case of a patient with severe hypoactive delirium on a background of Alzheimer's dementia with a significant lasting cognitive deficit. This case presents the devastating impact of delirium on the lives of patients and relatives. The need for more awareness of delirium amongst health care professionals, more routine risk assessment and more studies aimed at managing hospitalised patients with delirium is also implicated.

## 1. Introduction

Delirium is an acute and reversible condition causing disturbance of intellectual functioning, fluctuating consciousness and altered thinking. Cases typically resolve within four weeks and an underlying physical illness is frequently causative. The prevalence in the community ranges from 1%–2% and dementia increases the risk five fold[1]. Delirium is common in medical in-patients with a prevalence ranging from 10%–31% at admission and with the incidence of new cases per admission ranging from 3%–29%[2]. It is well documented that delirium has adverse physical, cognitive and psychological consequences and an association with increased mortality, increased length of stay and institutionalisation[2]. Whilst delirium has been consistently described since the second century[3], awareness amongst clinicians varies and evidence for interventional measures to improve outcomes is sparse. We describe a case of severe hypoactive delirium with no improvement demonstrated over a three month period and a

poor outcome.

## 2. Case report

A 71 year old lady of African descent was admitted to a large tertiary hospital following a seizure at home. Her past medical history was remarkable for hypertension, Alzheimer's dementia, for which she takes donepezil, and a meningioma, picked up incidentally during routine imaging. She was previously cared for at home by her son and her most recent standard mini mental state examination (SMMSE) prior to admission was recorded at 25 out of 30. She was known to drink 250 millilitres of whisky a day at home and did not smoke. She had a previous seizure one year prior to admission and following this, remained well controlled on valproate. She was initially treated for an alcohol withdrawal seizure and was planned for discharge home.

She acutely deteriorated over a weekend with reduced consciousness; no clear cause was found. Her initial blood tests, imaging and microbiology tests were unremarkable. She was seen by our team and we diagnosed a hypoactive delirium and suggested the clinical team continue their search for a cause. Her SMMSE was 0 out of 30 and she

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displayed fluctuating consciousness and was unable to verbally communicate. She received a course of intravenous antibiotics for sepsis with an unknown source. She had a persistently raised serum CRP of ~50. An opinion was sought from neurology and infectious disease consultants and many investigations were performed over the subsequent two months. Two CT head scans did not demonstrate any change from previous imaging. An EEG was normal. A CT scan of the abdomen, chest and pelvis did not detect any source of infection. A lumbar puncture did not detect any abnormality. Autoimmune and myeloma screens were negative. Four sets of blood cultures were negative. Tumour markers were normal. A serum ammonia assay was within normal parameters. Empirical treatment for viral encephalitis was commenced with intravenous acyclovir. Serum syphilis antibodies were positive; however, this was thought to be a false positive result. The serum and cerebral spinal fluid *Treponema pallidum* particle agglutination test was negative.

There had been no improvement in cognition during this period of three months. The clinical team made the decision to halt the search for causative factors and plan discharge. After liaison with the family, it was decided that the patient would be best cared for in a residential home.

### 3. Discussion

Despite many advances over the last few decades, delirium remains a significant problem in hospitalised patients with dementia. The most important approach towards caring for a patient with delirium is the identification and treatment of the underlying cause[4]. Patients must also be nursed in an appropriate environment with a reality orientation and MDT approach[4]. In this case, elucidating the underlying cause was a difficult task. The diagnosis of delirium was made one week following admission to hospital, despite scoring poorly on cognitive assessment on admission. The initial impression on admission was that of alcohol related seizures. Treatment was commenced, including oral benzodiazepines on a reducing regime. The patient became more unresponsive and this was attributed to over medication. The meningioma was also implicated, however, this lesion did not display any concerning features on imaging and was thought to be innocent. We advised stopping the anticholinesterase inhibitor temporarily as we noted that the patient had a prolonged QTc and these drugs can rarely cause seizures. The combined effect of a history of alcohol excess, benzodiazepine therapy, the anticholinesterase inhibitor and an incidental meningioma lead to the delay in diagnosis of a hypoactive delirium. The relationship between the duration of diagnostic delay of delirium and clinical outcomes is not documented in the literature; however, it is reasonable to assume an association with poorer outcomes.

Patients at high risk of developing delirium should be identified at admission with prevention strategies incorporated in their care plan[4]. A recent study proposes a

model for the predication of delirium in the intensive care unit[5]. Whilst no such model exists for older hospitalised patients, it is generally accepted that risk factors include old age, severe illness, dementia, physical frailty, dehydration, polypharmacy and alcohol excess amongst others. Thus, the patient should have been identified as high risk for developing delirium; however, routine risk identification is seldom employed.

A balance is to be had when trying to identify the causative factor for delirium. Whilst all efforts must be made to find the cause, two months of invasive investigation may cause distress for patients, relatives and carers. Best care would be a balance between intensive investigation and holistic management with guidance at all times from the family. In our case, there were regular discussions with the patient's son as her next of kin. All investigations were explained and justified and this led to a good working relationship with the family.

Perhaps the most striking finding from this case is the debilitating nature of delirium and the devastating impact it has on the lives of patients and relatives. We present a case of severe hypoactive delirium with little return of baseline functionality. Whilst health care professionals are becoming better at identifying patients with delirium, more education is needed for identifying patients at risk. More studies are also required to investigate potential pharmacological and non-pharmacological treatments.

### Conflict of interest statement

The authors declare that there are no conflicts of interest.

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